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CLINICAL ROENTGENOLOGY OF CARDIAC ENLARGEMENT

The heart is particularly well adapted to roentgenological investigation. It is a comparatively dense organ almost completely surrounded by radiolucent lung tissues. It is therefore possible to outline the contours and size of the heart and to examine each individual heart chamber for variations in size, shape and position, pulsations and density.

In this presentation only the usual methods of roentgenological examination will be discussed: *fluoroscopy* (roentgenoscopy), *teleroentgenography*, and *orthodiagraphy*. Accessory methods based on a more elaborate technique, such as angiocardiography and roentgenkymography, require more space than is available here.

Fluoroscopy (roentgenoscopy) is the direct examination of the patient behind the fluorescent screen. In spite of the exaggeration in size due to divergence of rays when the object is close to the roentgenray tube, this method is of definite value for estimating individual chamber size or the structure of the great vessels. It is inexact for measuring total heart size, but sufficiently accurate, as well as convenient and economical, to permit the diagnosis of cardiac enlargement as early as, or earlier than, the other usual methods.

The cardiac contours should be studied, not only in the postero-anterior position, but also in various degrees of rotation (usually right anterior oblique and left anterior oblique positions) so that depth also may be estimated. A description of the normal range of variations of the cardiac contours in the various positions is available in a publication of the American Heart Association[1] and in various other texts on the subject.

Teleroentgenography is the taking of chest roentgenograms at a standard distance of six feet or two meters. At such distance the divergent rays, delineating the borders of the heart shadow, exaggerate the true size to a minimal degree. Films taken in oblique positions may be taken at less than six foot distances without seriously detracting from the value of the method. Tables of normal measurements of the transverse diameter and of the surface area of the heart have been published. These are valuable as screening procedures, but are less useful, except for comparison with other serial roentgenograms, when applied to the individual case.

Orthodiagraphy utilizes central or parallel rays to outline the cardiac contours, enabling one to make a tracing of almost exactly same size and shape as the true outline of the heart in the plane under observation. This method is time consuming, the dangers of X-Ray exposure are increased, but in skilled hands actual error is slight. Transverse diameters may be measured or surface areas estimated and these may be compared with prediction formulae for orthodiagraphy. The disadvantage of this method, like that of prediction methods in teleroentgenography, is that it is fairly reliable only when definitely normal or definitely increased measurements are concerned. The borderline between normal and abnormal is not sharp, and figures greater than predicted are obtained not infrequently in normal individuals. Indeed, since a ten percent variation is not considered abnormal, the value of the measurement is obviously not great in the borderline group, where a decisive answer would be most desirable.

The exact estimation of total size when the heart is enlarged is of little value. It is of far greater importance to ascertain which chambers participate in the enlargement and to what degree, but such information cannot be obtained from measurements alone. From the practical point of view fluoroscopy is by far the most important of the roentgenological procedures because it permits one to estimate the size of individual heart chambers and great vessels and also to observe the amplitude and character of pulsations, intracardiac densities, and calcifications. On the other hand, roentgenograms permit much better definition of lung detail and so are often valuable in diagnosing the failing heart. Roentgenograms taken in the postero-anterior and oblique positions are useful as a permanent record and for comparison with subsequent examinations.

Criteria for estimating individual chamber enlargement are available.[1] In the following sections an attempt will be made to show how information concerning individual heart chamber size may be helpful in the diagnosis of some of the commonly encountered disorders of the heart. Note that the emphasis is put on the individual heart chambers rather than on the total

heart size or the cardiac disorder. This approach differs from one utilizing "typical configurations." Such configurations are often absent, and, even when present, assume clinical connotations which may be erroneous. For instance, a "mitral" shaped heart may be present secondary to chronic pulmonary disease or to thyrotoxicosis; the mere mention of "mitralization" may induce error. Estimation of the degree of individual chamber enlargement permits an evaluation of hemodynamic or physiological processes and, often, of the sequence of involvement. Such an estimate may correlate with the clinical diagnosis, but, if it does not correlate, a re-evaluation of the clinical and roentgenological factors is indicated.

Enlargement of a heart chamber is not specific for any clinical disorder. It is only the manifestation of the necessity of that chamber to hypertrophy and dilate secondary to disease and to the increased demands put upon it relative to its capacity to meet such needs. Chamber enlargements differ only quantitatively, not qualitatively. Moderate left ventricular enlargement is the same in size and shape in hypertension as it is in aortic or mitral insufficiency.

LEFT VENTRICLE. *No Enlargement:* The left ventricle is characteristically small in mitral stenosis; it is frequently displaced to the left by enlargement of the right ventricle, and posteriorly by the rotation of the heart on its vertical axis.

The left ventricle is not enlarged in uncomplicated arteriosclerotic heart disease (coronary sclerosis with angina pectoris), but may be enlarged slightly, moderately, or even markedly weeks or months after acute myocardial infarction, even in the absence of significant manifestations of heart failure.

Slight or Moderate Enlargement: Such enlargement is frequently present in hypertension, aortic insufficiency, aortic stenosis, and mitral insufficiency. In the transient or labile stages of hypertension the left ventricle is rarely enlarged. Persistent hypertension may lead to either rounding of the uper left ventricular contour without increase in chamber size (due to concentric hypertrophy) or to actual enlargement of the left ventricle. Elongation and dilatation of the ascending aorta, visualized best in the left anterior oblique position by an increased curvature and anterior projection of its contour, is much more frequent in hypertension than left ventricular enlargement or concentric hypertrophy.

In aortic insufficiency (syphilitic, rheumatic, or bicuspid aortic valve) all grades of left ventricular enlargement may be encountered. When the pulse pressure is normal, the left ventricle is usually not enlarged, and, if enlargement is present, another etiological factor for this condition should be sought. When the pulse pressure is high (except for that caused by the non-elastic rigid aorta of arteriosclerosis), the left ventricle is usually enlarged while the amplitude of left ventricular and aortic pulsations is considerably increased.

In aortic stenosis the left ventricle is usually only slightly or moderately enlarged. When massive enlargement occurs, it is likely to be due to an associated aortic insufficiency, coronary artery disease, or hypertension.

In mitral valvular insufficiency the left ventricle is only slightly or moderately enlarged. This grade of enlargement with a disproportionately greater degree of left atrial enlargement suggests or confirms the diagnosis.

Marked to Excessive Enlargement: The most common causes for such enlargement are aortic insufficiency, combined aortic stenosis and insufficiency, and hypertension. When massive left ventricular enlargement is present in the absence of the signs of valvular heart disease, antecedent hypertension is often postulated. This supposition may be confirmed by a more thorough search of the past records of the patient, by the demonstration of dilatation of the ascending aorta, or by finding arterial narrowing and arteriovenous compression when the fundi are examined.

Ventricular Aneurysm: Ventricular aneurysm is the common usage term applied to a localized bulge in the ventricular wall representing a healed or almost healed area of myocardial damage. The most frequent site is at the apex of the left ventricle, which should be studied early in deep inspiration. In late inspiration the Valsalva effects supervene and the bulge may diminish or even disappear. Other less common sites of bulging are high on the lateral wall and on the posterior wall. Examination in the oblique positions as well as in the postero-anterior is necessary. Increase in density due to layers of intraventricular (mural) thrombi, incisura, pericardial adhesions, and on rare occasions calcification may be helpful in the recognition of the affected site. Thinning of almost the entire lateral wall of the left ventricle occasionally occurs and is difficult to differentiate from massive left ventricular enlargement.

Paradoxical Pulsations: During systole the normally pulsating left ventricle moves inward at its upper and lateral contours and upward at its lower and inferior contours. Outward motion observed during systole or inward motion during diastole are abnormal and are termed paradoxical. Roentgenkymography (in particular electrokymography) is a graphic method useful for the demonstration of such pulsations.

Demonstration of paradoxical pulsations, when there is slight or no left ventricular enlargement, usually is highly suggestive of localized mycardial damage often with thinning of the left ventricular wall. The site of the paradoxical pulsations, however, does not necessarily correspond to the area of damage. Observation

of paradoxical pulsations may be important in establishing the diagnosis when the electrocardiographic evidence is not specific or when the clinical data are inconclusive.

Paradoxical pulsation associated with marked grades of left ventricular enlargement does not have the same significance as that associated with slight enlargement. It has on occasions been observed when there is no localized damage demonstrable grossly or microscopically.

LEFT ATRIUM. *No Enlargement:* The left atrium may not be enlarged with mitral stenosis, though, to be sure, absence of enlargement is unusual. Characteristically there is very slight or no enlargement in congenital heart disease (except in Lutembacher's syndrome and in patent ductus arteriosus), and in heart disease due to hypertension of the lesser circuit caused by pulmonary disease.

Slight to Moderate Enlargement: Such enlargement is characteristic in mitral valvular disease, where the enlargement is disproportionate to the size of the left ventricle. It is often present with left ventricular failure, in which case the grade of enlargement is less than that of the ventricle. Slight grades of left atrial enlargement are often present when paradoxical pulsations due to myocardial infarction are present even though the left ventricle is not demonstrably enlarged.

Marked to Excessive Enlargement: Giant left atria, appearing on the left as well as the right cardiac contours in the postero-anterior position, and extending far back into the retrocardiac space, and frequently elevating and compressing the left main bronchus, are characteristic of mitral valvular disease (almost always rheumatic, never with calcified annulus fibrosus).

Moderate to marked grades of left atrial enlargement may be secondary to left ventricular enlargement and left ventricular failure. In this situation the degree of atrial enlargement is usually relatively less than that of the ventricular enlargement.

RIGHT VENTRICLE. *Slight or Moderate Enlargement:* Dilatation of the trunk, the primary and secondary branches of the pulmonary artery, and of the outflow tract of the right ventricle is characteristic of pulmonary hypertension of moderate or long duration due to pulmonary disease, to mitral valvular disease, or to left ventricular failure.

Marked Enlargement: Frequently marked enlargement is an indication of rheumatic mitral valvular disease of long standing, with or without evidence of right ventricular failure (neck vein distension, hepatic enlargement, edema). In left ventricular failure marked enlargement is almost invariably associated with manifestations of right ventricular failure. In chronic pulmonary heart disease demonstration of inflow tract enlargement added to the previous outflow tract enlargement is practically always an indication of impending or past failure and is an ominous prognostic sign. Marked enlargement of the right ventricle may occur rapidly with pulmonary embolization and infarction, with manifestations of right ventricular failure being added to the acute signs of pulmonary distress.

RIGHT ATRIUM. *Slight or Moderate Enlargement:* Such enlargement is not uncommon in rheumatic heart disease in the absence of clinical manifestations of heart failure. In pulmonary heart disease and in left ventricular failure right auricular enlargement is almost invariably associated with current or past manifestations of right heart failure.

Marked Enlargement: Such grades of enlargement are common in rheumatic heart disease, either secondary to right ventricular failure or to organic tricuspid valvular involvement. Somewhat lesser grades of enlargement may be found in congenital heart disease with shunts of blood from the left to the right sides and with tricuspid stenosis or atresia. The roentgenological distinction between enlargement of the trabeculated portion of the right atrium (including the auricle proper) and the smooth portion between the orifices of the superior and inferior venae cavae is not too important since marked grades of right atrial enlargement may occur without involvement of the smooth posterior portion.

GENERALIZED CARDIAC ENLARGEMENT. In the majority of cases of organic heart disease more than one chamber is found to be enlarged (multiple chamber enlargement). Enlargement of all chambers is termed generalized chamber enlargement. When the enlargement of all the chambers is more or less symmetrical, a uniform or systemic agent is usually found to be the cause. Generalized chamber enlargement thus may be symmetrical or asymmetrical, and from the proportionate degree of enlargement of each of the chambers proper inferences may be drawn. Examples of asymmetrical generalized chamber enlargement are hypertensive and rheumatic valvular heart disease. Examples of symmetrical generalized cardiac enlargement are: primary or severe secondary anemias; myxedema (possibly with a pericardial effusion); thiamine deficiency; amyloid and glycogen storage disease; diffuse, focal, and interstitial myocarditis.

AORTA. Dilatation of the ascending aorta is frequent in clinically significant aortic insufficiency, hypertension, and syphilitic aortitis. In aortic insufficiency the left ventricle is also enlarged, and the amplitude of pulsations of both the aorta and the left ventricle is markedly increased. In hypertension of moderate to long duration the left ventricle often is enlarged, while the amplitude of aortic pulsations is within the normal range. In syphilitic aortitis the amplitude of aortic pulsation is within the normal range, while the left ventricle is not en-

larged unless there is an associated clinically significant aortic insufficiency.

Elongation and dilatation of the transverse portion of the aortic arch is secondary to the dilatation of the ascending portion, or it may be due to arteriosclerotic involvement which is more extensive in the abdominal and thoracic portions of the aorta than in the descending portion of the aortic arch. In the latter condition demonstration of calcific deposits in the aortic wall is relatively common; they should be looked for in oblique as well as in the postero-anterior positions. Calcification of the aortic wall involving only the ascending aorta is almost invariably associated with syphilitic aortitis.

The length of the transverse portion of the aortic arch often has a direct bearing on the demonstration of left atrial enlargement. In the middle and later years of life adhesions between the aorta and esophagus are usually fibrous in character in contrast to the areolar tissue type of attachment in youth. Because of these fibrous adhesions elongation of the transverse portion of the aortic arch may pull the esophagus away from its usual prevertebral position into the left costovertebral gutter, and away from its usually intimate relationship to the left atrium. Under these circumstances the use of a barium filled esophagus to demonstrate left atrial enlargement may lead to erroneous interpretation.

Demonstration of a dilated ascending aorta and a poorly visualized descending portion in children or young adults should be considered to be at least suggestive of coarctation of the aorta. Visualization of a dilated left subclavian artery plus erosion of the lower margin of some of the ribs (notching) is confirmatory evidence. If in addition the blood pressure in the antecubital space is elevated, or if it exceeds the blood pressure in the popliteal space, the diagnosis is established. Angiocardiography or aortography should be performed to demonstrate the extent and site of constriction, and the presence of collateral arteries and major arteries useful for anastomosis.

Left ventricular enlargement without aortic dilatation calls for consideration of some of the rarer causes for such a condition, such as subendocardial fibrosis, idiopathic myocarditis, absent or aberrant left coronary artery, and amyloid or glycogen storage disease. Another frequently unrecognized cause for this combination in young adults is myocardial infarction with vague onset of signs and symptoms and with equivocal electrocardiographic findings.

SUMMARY

1. Three common types of roentgenological methods of estimating heart size are presented.

2. The clinical importance of cardiac fluoroscopy is stressed.

3. An attempt is made to illustrate how roentgenologic information concerning the size of individual heart chambers contributes to the diagnosis of common disorders of the heart.

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REFERENCE

1. Nomenclature & Criteria for Diagnosis of Diseases of the Heart. Fourth Edition, sixth printing. New York Heart Association, 1946, New York, New York.